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RUPTURE OF MIDDLE MENINGEAL ARTERY WITHOUT FRACTURE;

LIGATION OF COMMON CAROTID ARTERY.

BY

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RUPTURE OF MIDDLE MENINGEAL ARTERY WITHOUT FRACTURE. LIGATURE OF COMMON CAROTID ARTERY FOR SECONDARY HEMORRHAGE.

BY JOSEPH RANSOHOFF, M.D., F.R.C.S. ENG.,
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ON March 12th, Joseph B., aged twenty-eight, merchant, fell from a ladder into a shaft. Height of fall, about eight feet. When assistance, which was close at hand, reached him, he was perfectly conscious, complaining only of severe pain in the forearm. Dr. Charles Kearns, who was summoned, recognized a Colles fracture, and after dressing it sent the patient to his home, a distance of two miles from the site of the accident. At no time during a full week from the date of the fall did the patient complain of headache, nausea, vomiting, or indeed anything indicative of head injury. He walked about the house and the streets, transacted some business; ate well, slept well, and appeared in his usual mental condition. On the morning of the eighth day, after a good night, he descended to the dining-room, complaining of some headache. A cup of coffee was taken but rejected. Seeking relief in sleep, the patient lay down upon a couch, where he at once lapsed into a condition of unconsciousness from which he could not be roused.

Three hours later I saw him, in consultation with Drs. Kearns, Nickles, and Forchheimer. His breathing was stertorous and 28 per minute; pulse 40 and full; temperature normal; coma profound; conjunctival and cutaneous reflexes entirely wanting; irregular twitching and tossing movements affected the face and extremities on both sides of the body.

There was neither mono- nor hemiplegia. The right pupil was small and sluggish; the left dilated almost at maximum and fixed.

The urine removed by catheterization had a specific gravity of 1040 and gave the sugar reaction to both Trommer's and Boettger's tests. A careful examination of the head failed to reveal evidences of trauma.

Although the clinical picture was that of cerebral compression from sub-cranial hemorrhage, and not that of diabetic coma, the possible presence of the latter condition could not be excluded. A drop of croton oil was therefore administered for its derivative effects.

During the five hours following the first examination, the pulse had risen to 70; the temperature to 101° in the rectum. The coma remained unchanged. Respiration stertorous as before. In expiration a slight paresis of the right cheek seemed discernible to one of the consultants. It certainly was not pronounced.

Recognizing in the increasing pulse-rate and rising temperature further evidences of hemorrhage and speedy dissolution, it was concluded to trephine over the left meningeal artery. When the scalp was shaved, it showed no trace of recent injury. It was rendered thoroughly aseptic and bandaged to prevent hemorrhage.

A semilunar flap, with base below, was then made, its centre being just two inches behind and on a level with the external angular process. On exposing a large portion of the temporal fossa, no trace of either fracture or fissure was seen. A bluish tinge of the squamons portion of the temporal bone gave certainty of a clot underneath. The removal of a small button of bone from this point was followed by a very copious flow of dark fluid blood, on the cessation of which a clot was exposed which was too firm to be removed through the small trephine opening. This was enlarged with rongeur forceps until it measured two inches in its antero-posterior and one inch in its vertical diameter. The firmness of the clot resisted irrigation. It had to be scooped out with the finger, and measured about six fluidounces. The anterior and posterior boundaries of the middle cranial fossa could be readily felt, the dura being separated by an interval of more than an inch from its bony covering.

With the removal of the clot all hemorrhage ceased. The cavity was irrigated with a mercuric solution, a small drainage-tube inserted, and the wound closed.

During the operation, which was made without an anæsthetic, the dilatation of the left pupil gradually subsided, so that at its end, it was little, if any, larger than the right. The pulse had risen rapidly, and when the patient was placed in bed was 140 and very feeble.

He appeared moribund. Six hours *post operationem* consciousness returned, the patient asking for water. In the morning, while inclined to delirium, he was rational most of the time, although quite restless. He recognized his attendants perfectly, and was able to converse in a rational manner. Pulse 114. Temperature normal. The urine removed by catheter showed a specific gravity of 1016 and was free from sugar.

The first dressing and the drainage-tube were removed on the third day, when there was a slight staining of the bandage. There was primary union of the entire wonnd, except where the drainage-tube had been. For a week thereafter the temperature did not rise over 99.5°; the pulse rate was reduced to 90; the delirium subsided, and the patient appeared on the road to recovery. On March 28th, nine days after the operation, while the patient was straining at stool, a profuse hemorrhage supervened, saturating the dressings and bed-linen. When Dr. Nickles, who was summoned, arrived, the hemorrhage had ceased. The dressings were changed, the sitting posture ordered to be maintained and applications of ice to the head.

March 29. Was called at 5 A. M. for recurrent hemorrhage. On freely opening the wound, which had firmly united, and removing a large coagulum, bright arterial blood welled from its depth. Plugging with iodoform gauze failed to check it.

In the hope of reaching its source, the aperture previously made was enlarged downward, but without avail. Plugging the cavity was again resorted to with negative results. Nothing remained but compression or ligation of the common or external carotid artery. The extreme restlessness of the patient precluded the former practice. The profuseness of the hemorrhage made it appear possible that its source was the internal carotid. The common carotid artery was therefore exposed and included in a catgut ligature at the point of election.

Unfortunately, as the result shows, proper precautions looking toward asepticism could not be taken. The operation had to be performed at once on the blood- and food-stained bed. The hemorrhage subsided as soon as the ligature was brought home. Fearing wound infection, both wounds were thoroughly irrigated and the cervical closed over a catgut strand, while the cranial wound was left open.

The patient rallied well from the loss of blood and the operation, but delirium returned and with it extreme restlessness and insomnia.

Increasing doses of morphine were needed to procure sleep. Sulfonal and the bromides were faithfully tried but found useless.

The first change of dressings was made forty-eight hours after the operation. Suppurative processes had been established in both wounds. On raising the scalp flap for irrigation the pulsations of the dura were plainly visible. The suppuration over the carotid appeared to be superficial. The local and general conditions could leave no question as to the existence of pyæmia. On the fifth day, with a sudden rise of temperature to 103.5° and increase of delirium, there developed an embolic pneumonia with profuse expectoration of blood, which continued for twenty-four hours and then gave way to purulent sputum. After continuing for four days this also subsided. On the tenth day the patient again seemed on the road to recovery. The temperature continued at about 100°; the pulse at about 108, and of fair volume. The delirium was subsiding and sleep was easily, though artificially, procured. Both wounds were covered with granulations and presented a healthy appearance.

On the fifteenth day hemorrhage supervened from the carotid at point of ligation, but subsided spontaneously for several hours. While discussing the advisability of treating it expectantly, a sudden gush of blood decided the course. With the assistance of Dr. Dandridge, the wound was rapidly opened and the bleeding vessel sought for. The ends were separated more than an inch. The bleeding came for the most part from the distal portion of the vessel and only with great difficulty could this be caught and retained in hæmostatic forceps. The hemorrhage was checked, but the patient, who had lost consciousness soon after the artery gave way, failed to rally. He died ten hours after all bleeding had ceased. A post-mortem could not be obtained.

If an apology be needed for offering the record of a case in many respects similar to the more than 400 cases recorded and tabulated by Vogt, Wiessman, Jacobson and others, I beg that you will find in it some of the unusual features preseuted. Of all the evidences of middle meningeal extravasation, the interval of lucidity between the accident and the appearance of compression symptoms comes first in importance. This interval may be only a few minutes, it often is one or two hours, but rarely as many days. Not once in twenty cases does the in-

terval of freedom from brain symptoms last longer than forty-eight hours. Koenig alludes briefly to a case in which *marked* cerebral symptoms developed on the eighth day, and in one of Wiessman's tables (No. 171)[1] coma supervened as late as the eleventh. But this was a case of extensive compound basal fracture in which the coma developed as a result of secondary hemorrhage.

In rather an extensive search of the literature of allied cases I have found none in which, as in the case reported, a full week passed before any symptoms of intra-cranial trauma developed.

The pressure of a firm clot, and of a large fluid extravasation, above the dura may explain the long delay of symptoms. Experiment and clinical observation abundantly demonstrate the ready adaptability of the brain to moderate general compression gradually applied. It is only when the limit is passed that this makes itself manifest. The firm clot was probably the result of a hemorrhage during the first day after the accident, whereas the fluid portion of the hæmatoma was recently extravasated, the bleeding becoming *foudroyante* when the recumbent posture was taken by the patient for the relief of headache. It is in many cases of meningeal hemorrhage as in idiopathic apoplexies: the severe symptoms of compression do not supervene until the patient has been placed in bed.

Another feature of the case reported, is the absence of paralytic or paretic symptoms on the opposite side of the body.

In 257 cases collected by Wiessman this absence was specially noted only 16 times.

In Jacobson's 70 cases[2] the absence of paralysis was noted in only 2.

In each case middle meningeal extravasation had taken place. In both there was much blood beneath the scalp, and therein lies the explanation of the absence of paralysis. The course of the artery over the face, arm- and leg-centres readily explains the frequency of contra-lateral paralysis in these cases. Though the effects of a localized compression are felt by the

[1] Wiessman: Zeitsch. f. Chir., Bd. xxi. S. 300.

[2] Jacobson: Guy's Hosp. Rep., 1886.

cerebrum as a whole, the parts in proximity are most affected. Therefore in hemorrhages which are partly basal the probability of paralysis of the opposite side diminishes; while that of direct pressure on the oculo-motor nerve and consequent dilatation of the pupil increases. Hutchinson[1] was the first to call attention to this condition of the pupil in two cases. Its diagnostic importance in the absence of other paretic symptoms can hardly be overrated. In the tabulated cases of Wiessman where dilatation of the pupil existed at all, it was present twenty times on the side of extravasation and only four times on the opposite. There need not be any concomitant paralysis of the extrinsic ocular muscles. Only four cases have been recorded in which there was either ptosis or paralysis of the external rectus.

In the case presented the dilatation of the pupil was the only evidence pointing to the side of the lesion, and in the absence of paralysis of the face or arm led me to believe that hemorrhage extended more to the base than is usually the case.

The trephine was accordingly applied half an inch below the point generally selected in cases of supra-dural hemorrhage. Without "Hutchinson's pupil," operative interference in this case would have been impossible, and its immediate disappearance on removal of the clot amounted to a physiological experiment.

Another feature of this case, to my knowledge not mentioned in any case hitherto reported, is the glycosuria, the relations of which to disturbed circulation of the intra-cranial viscera is more readily recognized than satisfactorily explained.

Quite recently Nagel[2] reports two cases in which the diabetes followed directly an apoplectic attack and in which it improved with the mitigation of the cerebral symptoms.

In spinal compression similar results are occasionally encountered. Baum[3] and Scheuplein[4] report cases of injury and disease in which the relief of compression of the cord was speedily followed by disappearance of the glycosuria.

[1] Hutchinson: Lond. Hosp. Rep., iv. p. 20.
[2] Nagel: Schmidt's Jahrb., 219, p. 475.
[3] Baum: Berl. klin. Wochensch., 1880, p. 608.
[4] Scheuplein: Arch. f. klin. Chir., Bd. xxix. S. 365.

In the case reported the urine was normal within twelve hours of the operation. Its subsidence with the disappearance of the coma, slowness of pulse, prove it to be but an evidence of compression.

Although deficient in post-mortem evidence, this I believe adds another to the small number of cases of supra-dural meningeal hemorrhage without fracture. Fully aware that the possibility of such an occurrence has often been denied, and that Marchant,[1] voicing the opinion of the latest French surgeons, rejects such lesions as "not proven," the evidence in this case appears to me sufficient to warrant the view expressed.

The greater portion of the temporal and part of the zygomatic fossa were exposed; there was no trace of fracture or fissure. Digital exploration of the middle fossa from its anterior to its posterior limits and almost to the foramen spinosum was also negative in its results. The subject was young, and the skull thin-walled Its elasticity surpassed the arterial resistance. As in other cases of this nature, the injuring force seemed so slight as to leave no evidence of damage even to the soft parts. There may, of course, have been a fissure of the base to the inner side of the point explored; but nothing in the clinical history warrants such an assumption.

There was no bleeding from the ear, no paralysis: and concussion symptoms, if at all present, were so slight as to form no factor in the record of the case.

Banner[2] reports a case in which the artery was torn across just within the cranium. This, I believe, occurred in my patient, the extravasation continuing until the more adherent parts of the dura along the borders of the fossa were reached. Following the classification of Kroenlein,[3] the case is one of circumscribed temporo-parietal hæmatoma.

Secondary hemorrhage is not often mentioned as a cause of death from rupture of the middle meningeal and its appearance so late as the eighth day, as in the case reported, is certainly

[1] Marchant: Thèse de Paris, 81, p. 27.
[2] Banner: Trans. Provincial Med. and Surg. Association, 1841.
[3] Kroenlein: Zeitsch. f. Chir., Bd. xxiii. S. 209.

uncommon. In cases of penetrating gunshot and stab wounds it has developed as late as the twelfth, the eighteenth, and twenty-first day. In the case reported by Gamgee[1] the hemorrhage sprang from a false aneurism; in the case of Lang,[2] the bleeding penetrated into the cerebrum through a punctured wound of the dura, and in the case of Alexander,[3] so far as I know the latest case of secondary hemorrhage from the meningeal recorded, the injury followed an extensive shell-fracture of the left temporal region. While these cases have only the secondary hemorrhage in common with the one reported, together they demonstrate that the danger from this source continues longer than the calibre of the vessel would lead one to suppose.

A more important problem than that of hæmostasis rarely presents itself for technical solution and this applies particularly to secondary hemorrhages where impassable anatomical bounds limit the field of operation. When the secondary bleeding presented itself as a thing to be speedily met there passed before me the many methods of securing the bleeding vessel.

Direct ligation of the vessel, inclusion with the transfixed dura and plugging its osseous canal were not attempted, on the principle that a man cannot be hanged until caught. When tamponing the cavity failed nothing remained but the ligation of the common carotid artery, a plan originally suggested by Forneaux Jordan[4] as in the first place preferable to trephining. Compression of the artery was successfully applied by Mr. Howse[5] for three hours for meningeal hemorrhage, but the comatose condition of the patient admitted such practice. In a very recent article Mr. Horsley[6] recommends it in the management of intra-cranial hemorrhages. In a restless patient, except in a hospital ward, the plan appears to me unfeasible.

[1] S. Gamgee: Lond. Lancet, 1875, i. p. 535.

[2] Lang: Weismann: loc. cit., p. 331.

[3] Alexander: Surg. Hist. War, i. p. 314.

[4] Jordan: Med. Times and Gazette, 1863, i. p. 314: also Surgical Enquiries, 2d Lond. Ed., 1880, p. 182.

[5] Howse: Jacobson's cases, No. 9.

[6] Horsley: Brit. Med. Journ., 1889, i. p. 457.

From a rather careful search of the literature of the subject, I find that there are only three cases on record in which the common carotid was tied for hemorrhage from the middle meningeal, the operators being Bentley,[1] Alexander, and Gamgee.

The case of Alexander, already referred to, was the only one followed by recovery. In Bentley's case a fatal hemorrhage recurred on the third day. Roser's suggestion of ligating the external carotid found only one follower in Mr. Howse. This case also proved fatal.

In conclusion, I would remark that although the immediate cause of death was hemorrhage, this was but a sequel of septic infection. I am consoled in the belief that it was unavoidable. It is not often that surgical emergencies arise in which we cannot obtain asepsis; but one disastrous result like this brings into strong relief how helpless we are without this greatest factor of surgical success.

[1] Bentley: Surg. Hist. War, i. p. 255.

DISCUSSION.

Dr. W. W. Keen, of Philadelphia.

I think that it is very fitting that this case should be recorded because of the peculiarities to which Dr. Ransohoff has called attention. It certainly is excessively rare to have intra-cranial hemorrhage going on to such an extent as in this case for eight days without any signs of its existence. I congratulate Dr. Ransohoff upon the acumen with which he made the diagnosis, with almost complete absence of symptoms. I should like to suggest that the better plan, as shown by the history of several cases of hemorrhage from the middle meningeal artery referred to, would have been to tie the main trunk low down, enlarging the opening in the skull, and passing a ligature by means of a curved needle under the vessel. In the absence of any knowledge of where the artery was ruptured, it might be difficult to do this. While the problem can be solved easier now than at the time of the operation, it seems to me that the rule should be to ligate the middle meningeal, rather than the carotid artery. My remarks apply, of course, to the primary and not to the secondary hemorrhage. If the artery could have been found and tied, it would have prevented the secondary hemorrhage. Statistics have shown that the main trunk is rarely ruptured as compared with its branches. It is possible that the artery could not have been seen, yet if it could have been found at the first trephining, it would have been better.

Dr. F. S. Dennis, of New York.

I have had a number of cases of meningeal hemorrhage, and my experience is that ligation of the common carotid is uncalled for because the hemorrhage can be arrested by other means. After removing the bone, I have found it impossible to catch the vessel with forceps. I have then taken a tenaculum and passed it under the dura and thus drew up the dura, and seized the vessel and tied it. In that way the necessity for tying the common carotid is obviated. With the vessel raised in this way it is possible to tie it in the curve of the tenaculum.

During this spring I trephined in one case without meeting with hemorrhage. There was paralysis on one side. I then incised the dura, but found no clot. I next incised the cerebral substance and went into the lateral ventricle and removed a clot the size of a walnut.

This is, I believe, the first case in which a clot was removed from the lateral ventricle. This was a case of apoplexy due to traumatism.

In regard to sugar in the urine, I have seen it quite often. It will probably be found in those cases where there is any irritation of the pons.

Dr. James McCann, of Pittsburg.

I infer from the history which has been given of the case by Dr. Ransohoff that the hemorrhage was complicated by a fracture of the base of the skull, and that the vessel from which the bleeding came had been lacerated at some deep and inaccessible point, either in the membranes, or within the brain itself; and if this inference is correct, the methods which have been suggested for securing the vessel would have been of little use.

It should never be a matter of great difficulty to control hemorrhage from the meningeal artery or its branches, if the bleeding point is visible. The late Professor Mütter, of Philadelphia, many years ago successfully accomplished this by plugging the torn end of the vessel with a piece of pine wood. There ought not to be much trouble encountered in twisting or ligating the vessel, or in applying forci-pressure to it. But when the blood wells up from a point far down toward the base of the skull, and the bleeding vessel cannot be seen or reached, the difficulties are greatly increased. Such cases are frequently associated with serious injury to the brain and its membranes. A case presenting many points of resemblance to Dr. Ransohoff's is reported by Dr. J. Collins Warren, in *The American Journal of Medical Sciences* for May, 1890. A youth, aged seventeen years, was thrown from a horse and at once became comatose. There was profuse bleeding from the nose and left ear, with some escape of brain matter from the left ear; but there was no wound and no external evidence of depression; the pupils were normal, and there was no paralysis. An exploratory incision revealed a fracture extending downward from the squamous portion of the temporal bone toward the base of the skull. A portion of the bone was removed by trephine, and some clots and a quantity of fluid blood evacuated, when it was seen that not only was the middle meningeal artery torn and the membranes of the brain ruptured, but that a portion of the anterior and middle lobes of the brain was extensively lacerated. The bleeding was easily controlled by pressure. Under a rigidly antiseptic treatment, with free drainage, this man ultimately recovered. In the management of

Dr. Ransohoff's case it would probably have been better practice to remove with trephine, or rongeur forceps, an area of the skull sufficiently large to expose the bleeding vessel, and to secure it at the bleeding point, rather than resort to ligation of the carotid artery.

Dr. J. R. Weist, of Richmond, Indiana.

I wish to mention a case which is *apropos*. A man, forty-five years of age, in a quarrel was struck by the fist of a companion. He did not fall, but went about his work for a time and then walked two miles and commenced an action against the man who had struck him. He attended to some other business, and was about for four hours without special complaint. He then had severe pains in the head, and went to the office of a homœopathic practitioner. The doctor proceeded to bleed him, and about the time he had finished the operation the patient was seized with a violent convulsion. The convulsion was followed by coma. When I saw him he was profoundly comatose, with the hands contracted—equally so, as far as I could determine. The right pupil was slightly dilated. The left side of the head had been struck just above the middle of the parietal bone. At that point there was slight swelling and other evidences of contusion. It was clear that there was cerebral hemorrhage. I was unable to get any history of the convulsions beginning on one side before appearing on the other. Not much weight was attached to the difference in the size of the pupils for the reason that it seemed most probable the bleeding was on the side of the injury. I proceeded to trephine at the point of contusion. There were no signs of injury to the skull or membranes at that point. I at once trephined on the opposite side and found an extensive intra-dural hemorrhage. The clots were removed with the finger. I was unable to determine the point from which the blood came. Compression was made on the common carotid and continued some three hours. The patient never rallied from the coma, and died in six hours. The difficulty in this case before operation was to determine on which side the hemorrhage had occurred. If it is true that dilatation of the pupil indicates the side on which the hemorrhage is present, it is a valuable sign. If a prosecution for murder had followed in the case narrated, it is certain the question would have been raised if it were possible for the surgeon to determine the location of the hemorrhage before using the trephine.

Dr. Ransohoff.—I stated distinctly that when the secondary hemorrhage supervened, my first effort was to find the middle meningeal artery. I enlarged the trephine orifice as far as I possibly could, but the blood still seemed to come from the depth of the wound; I could see nothing of the artery. There was only one of two things to do: either to compress the carotid for a considerable length of time, or to tie the artery. Compression in this case was out of the question. The most interesting feature of the case is that the patient was perfectly well for eight days. Another fact of almost equal interest was the absence of fracture or other injury.